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Office of Administrative Law Judges
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**Issue date: 25Sep2001**

CASE NO.: 2001-BLA-334

In the Matter of:

KATHRYN L. CORNETT, SURVIVOR OF
ELMER R. CORNETT
Claimant

v.

CONSOL, INC.
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Respondent

APPEARANCES::

Thomas E. Johnson, Esquire
For the Claimant

William S. Mattingly, Esquire
For the Employer

Before: ROBERT J. LESNICK
Administrative Law Judge

## DECISION AND ORDER - AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901, *et seq.* (hereafter "the Act"). The pertinent implementing regulations appear at Parts 718 and 725 of Title 20 of the Code of Federal Regulations.

Benefits are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of coal miners who died from pneumoconiosis. *See* 20 C.F.R. § 725.1(a). Pneumoconiosis, commonly known as "black lung disease," is a chronic disease of the lungs and its sequelae (including respiratory and pulmonary impairments) resulting from coal mine employment. *See* 20 C.F.R. § 725.101(a)(20). *See also* 20 C.F.R. Part 718.

Following notice to all interested parties, a formal hearing was held before the undersigned on June 6, 2001, in Wheeling, West Virginia, in accordance with pertinent portions of 20 C.F.R. Part 725 and 29 C.F.R. Part 18. The record consists of the hearing transcript, Director's Exhibits Nos. 1 through 47, Administrative Law Judge Exhibit No. 1, Claimant's Exhibits Nos. 1 through 6, and Employer's Exhibits 1 through 12.[1]

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented.[2] Where pertinent, I have made credibility determinations concerning the evidence.

## Statement of the Case

On December 14, 1979, Elmer Cornett ("miner"), filed an application for benefits under the Act. (DX 28). A denial of the claim was issued by the Office of Workers' Compensation Programs ("OWCP") on April 22, 1980. (DX 28). The claim was not pursued any further by the miner. Accordingly, it is deemed that the miner's 1979 claim is administratively closed.

Mr. Cornett passed away on May 16, 1998. (DX 5 & TR 34). Kathryn L. Cornett, ("claimant") filed a claim for survivor's benefits on October 14, 1998. (DX 1). Employer was notified of the pending claim on March 24, 1999, and filed a timely controversion on April 8, 1999. (DX 11 & 14). A Notice of Initial Findings was issued by OWCP on March 26, 1999 finding employer to be the properly designated responsible operator and finding claimant entitled to benefits under the Act. (DX 12). The District Director ("Director") issued an Initial Determination finding claimant entitled to benefits from May 1, 1998 and that employer should begin payments. (DX 17). Employer requested a reconsideration of the Director's initial determination. (DX 19). The claim was subsequently denied by OWCP on October 27, 1999 because the evidence did not establish that coal workers' pneumoconiosis had caused the miner's death. (DX 23).

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[1] The following abbreviations have been used in this opinion: DX = Director's exhibits; EX = Employer's exhibits; CX = Claimant's exhibits; ALJX = Court exhibits; TR = Hearing Transcript.

[2] Employer submitted EX 9 through 12 under cover letter dated July 18, 2001. No objection to the admission of these exhibits was received. Therefore, they have been made a part of the record in this claim.

Claimant promptly requested that the claim be referred to the Office of Administrative Law Judges ("OALJ") for a formal hearing. (DX 24). The claim was referred to the OALJ on November 30, 1999. (DX 25). There appears to be some inconsistency in the administrative record in this claim. There is an additional referral letter in the record dated January 10, 2000. (DX 29). The claim was then remanded to the Director for the development of further evidence on March 9, 2000. (DX 30).

On May 3, 2000, the claim was denied by OWCP. Claimant was permitted to submit additional evidence after this denial. On October 27, 1999, the Director issued a Proposed Decision and Order Denying the Request for Modification. The Director determined that the additionally submitted evidence did not change the weight of the evidence, and that claimant failed to establish that the miner's death was due to coal workers' pneumoconiosis. (DX 42). Claimant requested that the claim be referred to the OALJ on November 15, 2000. (DX 44). The claim was referred to the OALJ on January 2, 2001. (DX 46).

The remaining contested issues are whether employer is the properly designated responsible operator and whether the miner's death was due to pneumoconiosis, as provided in the Act and applicable regulations. (TR 30).

## Applicable Regulations

Since claimant filed her application for survivor's benefits after January 1, 1982, this matter must be determined under Part 718 of the regulations, as amended. A survivor is automatically entitled to benefits as a result of a claim filed prior to January 1, 1982. However, a survivor is no longer automatically entitled to such benefits under a claim filed on or after January 1, 1982 where the miner is not entitled to benefits as a result of the miner's claim filed prior to January 1, 1982 or where no miner's claim was filed prior to January 1, 1982. *Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). In addition, the survivor is not entitled to the use of lay evidence, or the presumptions at §§ 718.303 and 718.305 to aid in establishing entitlement to survivor's benefits.

## Findings of Fact and Conclusions of Law

### Background

The miner, Elmer Cornett, was born on January 1, 1929. (DX 1). He married the claimant, Kathryn on July 15, 1972. (DX 6 & TR 34). The miner died on May 16, 1998. (DX 5). Claimant has no dependents for purposes of augmentation of benefits under the Act. (DX 1).

Employer concedes that the miner had 40 years of coal mine employment. (TR 29). I find that this determination is well supported by the evidence contained in the record. Thus, I find that the miner had 40 years of coal mine employment.

Claimant testified at the time of the hearing that the miner never smoked cigarettes to her knowledge. (TR 35). She met the miner between 1950 and 1960. (TR 35). Claimant testified further that the miner was 62 years of age when he became a federal mine inspector. (TR 35). Claimant also stated that the miner left coal mine employment before he was eligible for pension benefits because of his "breathing problems." (TR 36). Claimant further stated that the miner worked at the "seam" of the coal mine. (TR 37). Additionally, claimant testified that the miner had knee replacement surgery and also suffered multiple strokes in the last years of his life. (TR 36 & 38.

## Newly Submitted Medical Evidence[3]

*Death Certificate*

The miner died on May 16, 1998. (DX 5). The death certificate, certified by Dr. Stephen C. Ulrich, lists the primary cause of death as pulmonary failure with the underlying causes of stroke and multi-infarct dementia

*Autopsy Report*

An autopsy was conducted on May 16, 1998 by Dr. Katherine Tabatowski. (DX 7). The autopsy was limited to the thorax. Dr. Tabatowski noted the "presence of dust macules with associated mild centroacinar emphysema ... consistent with coal workers' pneumoconiosis" when taking into consideration the miner's employment history and the lack of smoking history.

Dr. Tabatowski conducted a gross examination of the organs, noting that the pleural surfaces of the lung were mottled black and pink. Additionally, Dr. Tabatowski noted that the lungs appeared to be well aerated with a few "palpable small nodules" being present. Dr. Tabatowski also noted that the cut surfaces of the lungs showed black reticulation and macular pigment deposition. Dr. Tabatowski also noted that the lungs exhibited "[s]cattered tiny barely perceptible nodular areas" present on sectioning with a "5 mm calcified black nodule in the right lower lobe."

Dr. Tabatowski explained her microscopic findings as follows.

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[3] While this decision must be based on a review and consideration of the administrative record as a whole, including the evidence submitted with the miner's claims, the exhibits from those claims will not be set forth except where it is important for an analysis of the issues in this survivor's claim. *See generally Wheeler v. Apfel*, 224 F.3d 891, 895 n. 3 (8th Cir. 2000); *cf. Fargnoli v. Halter*, 247 F.3d 34 (3d Cir. 2001) (ALJ not expected to make reference to every relevant treatment note from voluminous medical records in Social Security case).

> Sections of both lower lobes and right middle lobe reveals areas of acute pneumonia, in which alveolar spaces are filled by neutrophils, accompanied by congestion and hemorrhage. Microthrombi are present in a few of the pulmonary vessels. There is a slight degree of focal congestion and edema as well, in the lower lobes. Scattered deposits of anthracotic pigment are present throughout the lung sections and in the pleura; these macular and measure up to 4-5 mm in greatest dimension. Some of the deposits are associated with a slight degree of fibrosis, as demonstrated by trichrome stains. Sections of the right lower lobe reveal fibrotic and calcified granulomas. Fungal and acid fast stains are negative.

These findings lead Dr. Tabatowski to the following final diagnoses: dust macules with associated centroacinar emphysema consistent with coal workers' pneumoconiosis; acute broncho-pneumonia, bilateral lower lobes and right middle lobe; calcified and fibrocaseous granulomas, lungs and hilar lymph nodes; microscopic pulmonary thromoboemboli; mild coronary artherssclerosis; and status post cerebrovascular accident.

*Medical Documentation*

*Dr. Jeffrey A. Kahn*

Dr. Jeffrey A. Kahn conducted a NIOSH autopsy consultation report in this claim on October 14, 1998. (DX 8). Dr. Kahn is board certified in anatomical and clinical pathology.[4] (EX 4). Dr. Kahn stated that his microscopic examination of the miner's slides showed broad areas of pneumonia, and a moderate amount of coal dust present. Additionally, Dr. Kahn noted that coal dust was also present in the "walls of the terminal respiratory units where there is associated fibrous proliferation forming 'coal macules,' the hallmark lesion of coal workers' pneumoconiosis."

Dr. Kahn determined that the aforementioned macules involved up to 30% of the miner's terminal respiratory units. Dr. Kahn notes that these macules were present in all but one of the lobes of the miner's lung. Dr. Kahn also states that there are a "few small coal nodules" present that are representative of a more advanced stage of coal workers' pneumoconiosis. Dr. Kahn did not find any evidence of progressive massive fibrosis, but did find mild to moderate pulmonary emphysema. Dr.

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[4] Dr. Kahn is also the director of several pathology labs as well as serving as a clinical associate professor of pathology at the West Virginia University. Dr. Kahn holds several other academic appointments and has published numerous journal articles. Dr. Kahn is a consulting physician for NIOSH and has been for the last 12 years. In that capacity, Dr. Kahn has examined "tens of thousands" of lung slides to determine the presence of pneumoconiosis. *See* EX 4. Dr. Kahn is also involved in an ongoing study for NIOSH.

Kahn diagnosed the miner as suffering from moderately severe coal workers' pneumoconiosis, pulmonary emphysema, and acute pneumonia.

Dr. Kahn issued an additional report in this claim on August 7, 2000. (DX 40). At that time, Dr. Kahn reviewed what constituted the entire record at that point in the proceedings. Additionally, Dr. Kahn reviewed 19 microscopic slides created at the time of the miner's autopsy. In five of the slides, Dr. Kahn determined that the slides exhibited the same features as those described in his initial report. Dr. Kahn also pointed out the difference between coal macules and coal nodules. Dr. Kahn defined coal macules as the presence of coal dust in addition to the presence of fibrosis. A coal nodule is defined as "more advanced lesions with the same histologic components" and the presence of focal emphysema.

Dr. Kahn also reviewed 14 additional slides for this report. Dr. Kahn stated that these slides showed findings consistent with his initial report. Dr. Kahn noted that the slides were not of good quality. In one set of the slides, Dr. Kahn found that approximately 50% of the terminal respiratory units were involved with coal macules and small coal nodules. However, Dr. Kahn found that another set showed 20% of the terminal respiratory units involved.

Dr. Kahn took the time to explain focal emphysema and its relationship to coal dust inhalation. Dr. Kahn explained that focal emphysema involves the "expansion and distortion of the lumina of terminal respiratory units when a macule is formed." Dr. Kahn stated that he believes that coal dust inhalation contributes in the development of emphysema and may also lead to the production of bronchitis. Dr. Kahn also explained the mechanics of a death involving pneumonia.

In speaking of the miner directly, Dr. Kahn noted that "broad areas" of the miner's lungs were involved with the disease process. Dr. Kahn opines that pneumonia is more life threatening if a chronic lung disease is also present. This is so because the chronic lung disease "deprives the lung of functional reserve and impairs the ability to overcome insults." Dr. Kahn concluded that the miner's lung exhibited coal macules and coal nodules formed as part of the coal workers' pneumoconiosis process, and these macules and nodules distorted the miner's "pulmonary architecture" and disturbed the miner's air flow. As a result, the miner was disposed to the development of pneumonia.

Dr. Kahn opined that the miner's "emphysema, chronic bronchitis, coal workers' pneumoconiosis, and neurologic disease" all contributed to the miner developing pneumonia. Dr. Kahn concluded that each of these conditions "produced pathopsyiologic effects that aggravated the other diseases present and effects of the diseases contributed to the miner's death." Dr. Kahn concluded that "coal dust related chronic respiratory disease contributed to and hastened the [miner's] death."

Dr. Kahn was deposed in connection with this claim on December 20, 2000. (EX 4). Dr. Kahn began the deposition by explaining his understanding of coal workers' pneumoconiosis and its effect on the body system. Dr. Kahn explained that the presence of black pigment is insufficient to

diagnose coal workers' pneumoconiosis. Dr. Kahn also explained that when pneumoconiosis is present, it usually contributes to the miner's death. However, Dr. Kahn clarified that statement by saying that if the pneumoconiosis is "extremely minor" it does not usually contribute to death.

Dr. Kahn explained that in rendering his opinion, he assumed that the slides that he received were representative of the miner's entire lung. Dr. Kahn based this assumption on the fact that pathologists usually try to sample areas that represent the entire lung as well as the fact that the autopsy protocol states that each lobe of the lung was sampled. Dr. Kahn explained that there is no indication in the autopsy protocol that the prosector took samples from only the diseased areas. Dr. Kahn noted that the findings at autopsy were consistent with a finding of coal workers' pneumoconiosis. Dr. Kahn stated that the cause of the miner's death was "interaction of acute and chronic disease processes."

Dr. Kahn found focal emphysema that is consistent with coal workers' pneumoconiosis as well as either panacinar or centrolobular emphysema, both of which are more general types of emphysema. Dr. Kahn opined that the more general emphysema was likely caused by a combination of factors that includes coal dust inhalation. Dr. Kahn believes that emphysema also contributed to the miner's death because it hampered the miner's air flow.

Dr. Kahn opines that the multiple strokes suffered by claimant did not contribute to the destruction of the miner's terminal respiratory units. Dr. Kahn also explained that strokes do not cause emphysema. Dr. Kahn stated that there is no reference in the Bethesda Hospital medical records that the miner's stroke was effecting his respiratory function. Dr. Kahn states that the miner would not have suffered from shortness of breath at rest, but that the miner did suffer from a compromise of his pulmonary reserve.

Dr. Kahn testified that he also found pneumonia to be present. Dr. Kahn opined that, based on his clinical experience and fundamental medical knowledge, pneumonia occurs with more frequency when a person is afflicted with a chronic lung disease. Dr. Kahn stated that coal workers' pneumoconiosis "predisposes" an individual to the "microorganisms that produce the pneumonia infection." This happens because pneumoconiosis alters the air flow patterns and because of that alteration, there is less oxygen available to exchange and be picked up by the red blood cells to be carried to the rest of the body. Dr. Kahn opined that when the body is unable to oxygenate the blood and remove gaseous waste products that multi-symptom organ failure occurs. Dr. Kahn concluded that a person who is not afflicted with coal workers' pneumoconiosis would be in a better position to fight off pneumonia.

Dr. Kahn also addressed the miner's dementia and neurologic disease. Dr. Kahn states that these may have been contributing factors to the miner's death. However, Dr. Kahn states that coal workers' pneumoconiosis contributed to the death of the miner. Dr. Kahn states further that the severity of the miner's pneumoconiosis would be a hastening factor in the miner's respiratory death. Dr. Kahn also explained that the miner would have been expected to suffer from hypoxia (a decreased concentration of oxygen) with his coal workers' pneumoconiosis when he developed the pneumonia.

Dr. Kahn explained that when pneumoconiosis progresses to the point that nodules are present, there is a loss in pulmonary reserve. This can lead to greater degrees of impairment based on the extent of the disease present. Dr. Kahn opines that because of the amount of the lung involved with pneumoconiosis, the miner's lung could not function properly. Dr. Kahn states that he is able to render his opinion as to the functional significance of the miner's coal workers' pneumoconiosis because of the destruction by coal macules of the terminal respiratory units.

Dr. Kahn concludes that, taking into consideration all of the miner's chronic respiratory conditions, the miner would be unable to maintain employment in which the miner was required to complete manual labor.

*Dr. Stephen Ulrich*

Dr. Stephen Ulrich issued a report in this claim on November 6, 1998.[5] (DX 9). Dr. Ulrich is board certified in family medicine. Dr. Ulrich is an associate volunteer instructor at the Ohio State University and the Ohio University College of Osteopathic Medicine. Dr. Ulrich determined that the miner suffered from pneumoconiosis based on the miner's complaint of shortness of breath. Dr. Ulrich opines that coal workers' pneumoconiosis contributed to the miner's death in that the miner had an inadequate pulmonary reserve to compensate for the effects of the stroke suffered by the miner. Dr. Ulrich concludes that had the miner had "normal lungs," the miner would have lived longer.

Dr. Ulrich was also deposed in connection with this claim. (DX 31). Dr. Ulrich testified that he had been treating the miner periodically since March, 1985. Dr. Ulrich explained that the miner was reluctant to seek medical treatment. Dr. Ulrich further explained that the miner was being treated for both Sundowner Syndrome and dementia.[6] In April, 1998, Dr. Ulrich states that the miner suffered from aspiration pneumonia. This, Dr. Ulrich explains, is pneumonia that is a result of the "presence of fluids, food or other foreign objects that get into the lungs through the mouth or throat." Dr. Ulrich states that this is a common condition with people who have suffered a stroke. Dr. Ulrich opines that the aspiration was probably a result of the stroke, but that this condition can be seen in people who suffer from progressive dementia.

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[5] In addition to Dr. Ulrich's opinion, records from Good Samaritan Hospital are also included in DX 9. The notes of those records will be included in the section allotted for the documentation from that hospital.

[6] Dr. Ulrich explained that the difference between the two conditions is that Sundowner Syndrome causes confusion and disorientation after sunset. A person afflicted with this condition lacks visual stimuli after the sun sets and leads to the confusion. Dementia, on the other hand, is a "more global loss of function."

Dr. Ulrich states that the miner's immediate cause of death was pneumonia. Dr. Ulrich states further that with the aspiration pneumonia suffered by the miner, that there would be shortness of breath and the inability to get oxygen to the miner's brain and other vital organs. This inability to transfer oxygen is what Dr. Ulrich believes killed the miner. Dr. Ulrich opines that the miner died more quickly from the aspiration pneumonia because of the pre-existing pneumoconiosis condition. Coal workers' pneumoconiosis had weakened the lungs and had a detrimental impact on the miner's pulmonary reserve.

Dr. Ulrich explained that he thought that he had listed exposure to coal dust as a contributing factor on the miner's death certificate. However, no such designation appears on the certificate. Dr. Ulrich stated that, in treating the miner over the years, Dr. Ulrich believed that the miner suffered from a significant lung disease. Dr. Ulrich believes that this fact was overshadowed in the treatment notations because of the miner's stroke and high blood pressure. Dr. Ulrich opines that the miner's pulmonary condition had not been properly evaluated due to the miner's reluctance to be seen by physicians.

Dr. Ulrich states that there is not sufficient documentation to support a diagnosis of chronic obstructive pulmonary disease. However, Dr. Ulrich is confident, based on his experience with the miner, that the miner suffered from some degree of lung disease. Dr. Ulrich states that the miner's pulmonary conditions were not well documented because most of the miner's visits to physicians were for acute problems (stroke and alcohol addiction) that overshadowed the underlying pulmonary conditions.

Dr. Ulrich believes that the miner's pulmonary abnormalities were not a result of the weakness that the miner suffered from due to his prior stroke. Dr. Ulrich did not diagnose the miner as suffering from coal workers' pneumoconiosis during the miner's life. Dr. Ulrich did find that the chest x-rays taken during the miner's lifetime showed opacites consistent with coal workers' pneumoconiosis.

Dr. Ulrich concluded that coal workers' pneumoconiosis contributed to the miner's death. Dr. Ulrich explains that the disease contributed because the miner did not have an adequate pulmonary reserve to compensate for the weakening that resulted from the previous stroke. Dr. Ulrich opines that if a person's pulmonary condition is already weakened, that person is less likely to survive a bout with pneumonia. Dr. Ulrich opines further that had the miner had healthy lungs, he likely would have survived the pneumonia. Dr. Ulrich believes that the miner had enough already existing lung damage that coal workers' pneumoconiosis hastened the miner's death. Dr. Ulrich bases this opinion on his observation of the miner's abnormal breath sounds over the 13 years that Dr. Ulrich treated the miner and the shortness of breath that Dr. Ulrich observed that worsened just before the miner's demise.

*Dr. Joseph Tomashefski, Jr.*

Dr. Joseph Tomashefski, Jr. issued a report in this claim on August 13, 1999. (DX 21). Dr. Tomashefski reviewed the medical records in this claim as well as the pathology slides. Dr.

Tomashefski is board certified in anatomical and clinical pathology.[7] Dr. Tomashefski noted that the miner had 20 years of coal mine employment and 22 years as a federal mine inspector.

Microscopically, Dr. Tomashefski found "multiple 1 to 2 mm size peribronchiolar, pigmented macules containing course black particles and polarizable birefringent crystals." Dr. Tomashefski found that the macules present constituted 10% of the lung parencymal area and focal emphysema constituted 20% of the same area. Dr. Tomashefski found that the underlying cause of death was cerebral infarction and multi-infarct dementia, with the immediate cause of death being bronchopneumonia. Dr. Tomashefski opines that the neurologic disorders suffered by the miner predisposed the miner to episodes of aspiration pneumonia.

Dr. Tomashefski found that the miner suffered from "moderately severe" simple coal workers' pneumoconiosis. Dr. Tomashefski also found a more mild level of panacinar emphysema. This emphysema, according to Dr. Tomashefski was not related to coal dust exposure and is merely a condition of aging. Dr. Tomashefski opines that the miner's pneumoconiosis was not sufficiently severe to cause dyspnea on exertion and that the miner's terminal respiratory failure was a result of the acute bronchopneumonia and multiple pulmonary emboli.

Dr. Tomashefski found that the coal workers' pneumoconiosis present would have kept the miner from performing strenuous physical labor. However, Dr. Tomashefski found that the simple coal workers' pneumoconiosis present did not contribute to the miner's death. Dr. Tomashefski found that the miner was terminal due to the multi-infarct dementia and stroke. Dr. Tomashefski concluded that the acute pneumonia and pulmonary emboli alone would have been sufficient to cause the miner's death regardless of the presence of coal workers' pneumoconiosis.

Dr. Tomashefski was also deposed in the above-captioned claim. (EX 6). Dr. Tomashefski explained that simple coal workers' pneumoconiosis "rarely" causes symptoms and "almost never" causes death. However, if the pneumoconiosis is severe, Dr. Tomashefski believes that the condition can contribute to death. Dr. Tomashefski further explained that once a miner retires, simple coal workers' pneumoconiosis does not progress.

Dr. Tomashefski discussed the miner's history of cerebral vascular disease. Dr. Tomashefski explained that this caused "small areas of cell death" that caused destruction to a large part of the miner's brain. This, in turn, destroyed the miner's mental capacity and the ability of his body to function normally. Dr. Tomashefski also discussed the miner's pneumonia.

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[7] Dr. Tomashefski is a staff pathologist and professor of pathology at Case Western Reserve University. Dr. Tomashefski has multiple teaching responsibilities and has had multiple articles and text book chapters published. Dr. Tomashefski also is a member of several editorial review boards.

Dr. Tomashefski explained that pneumonia is caused by bacteria and the result is that the person suffers a decrease in lung function. Additionally, each time a person contracts pneumonia, the overall lung function is weakened. Dr. Tomashefski stated that when pneumonia is recurrent and related to vascular disease, that it is directly related to the problems the person encounters with swallowing. This difficulty in swallowing leads to the aspiration of bacteria into the lungs which causes pneumonia. Dr. Tomashefski opined that pneumonia is not caused by the inhalation of coal dust, but if the person suffered from a severe coal dust induced disease, that condition could aggravate the pneumonia. Simple pneumoconiosis would not impact the pneumonia.

Dr. Tomashefski discussed the autopsy report of Dr. Tabatowski. Dr. Tomashefski stated that he is unsure of Dr. Tabatowski's use of the phrase "black reticulation and macular pigment deposition." Dr. Tomashefski also discussed pleural adhesions. Dr. Tomashefski explained that these pleural adhesions are "bits of membranous connective tissue which adheres the parietal to the visceral pleural or the pleural lining of the lung to the pleural lining of the thoracic cavity." The adhesion is usually associated with an inflammatory process that had previously occurred in the pleura and healed. Dr. Tomashefski opines that the pleural adhesions found in the miner were due to the pneumonia and had no relationship to the inhalation of coal dust.

Dr. Tomashefski also discussed the black calcified nodule found in the lower right lobe of the miner. Dr. Tomashefski found that this nodule was not related to coal dust exposure, but rather it was caused by prior infections. Dr. Tomashefski bases this determination on the fact that coal workers' pneumoconiosis is usually found in the upper lobes.

Dr. Tomashefski also reviewed the lung tissue slides in this claim. Microscopically, Dr. Tomashefski found that he was able to see the distortion of the lung tissue at a very low magnification, but to identify the content of the macule, a higher magnification was necessary. Dr. Tomashefski found "discreet, coarse black particles within the macular lesion" and "birefringent crystals with polarized light." Dr. Tomashefski further explained that abnormalities associated with coal dust exposure usually deposit in the vicinity of the small airways.

Dr. Tomashefski also found areas of acute neutrophilic exudate. Finding neutrophils in the alveolar space, Dr. Tomashefski explains, "suggests that the body is mounting a response to bacteria and [this] process [in the lung] is known as bronchopneumonia." Dr. Tomashefski found areas of focal emphysema in multiple areas on the lung tissue slides. Dr. Tomashefski also noted that the miner was "throwing clots [i.e. pulmonary emboli] from other parts of his body into the pulmonary arterial circuit to obstruct the small pulmonary arteries." Dr. Tomashefski stated that these clots and the pneumonia together would cause a substantial decrease in oxygen. Dr. Tomashefski made a point to note that the pulmonary emboli are in no way related to coal dust exposure.

Dr. Tomashefski opined that the miner's death was caused by cerebral infarction and the disease that arose from that condition with the immediate cause of death being bronchopneumonia. Dr.

Tomashefski stated that the bronchopneumonia arose out of the miner's aspiration pneumonia which was precipitated by the cerebral infarction. Dr. Tomashefski characterized the miner's coal workers' pneumoconiosis as being moderate to severe. However, Dr. Tomashefski concluded that the condition did not contribute to nor hasten the miner's death.

Dr. Tomashefski opined that the miner's coal workers' pneumoconiosis may have been sufficient to cause shortness of breath but that such a conclusion would be speculative considering the lack of test results for lung function. Dr. Tomashefski opined that the miner's impairment caused by coal workers' pneumoconiosis would have been mild. Dr. Tomashefski bases this on the fact that the level of coal workers' pneumoconiosis found in the lungs of the miner would not have impaired blood oxygenation, however, the miner might not be able to conduct heavy manual labor.

Dr. Tomashefski also pointed out that pathologists are more likely to sample the diseased areas of the lungs, rather than taking a representative sample. Dr. Tomashefski opined that this usually leads to the diseased portions of the lungs being overrepresented in the slides. Dr. Tomashefski concluded that "no coal dust induced lung disease caused, contributed to nor hastened death" and that the miner "would have died at the same time and in the same way if he did not have coal workers' pneumoconiosis."

Dr. Tomashefski also issued a supplemental report in this claim on January 12, 2000. (EX 7). At that time, Dr. Tomashefski reviewed an additional 20 slides. Dr. Tomashefski stated that the slides presented features similar to the conclusions reached in the first report, but that the extent of the coal workers' pneumoconiosis present was less than that reported in the previous report. Dr. Tomashefski reported that the coal macules "comprise less than 5% of the parencymal tissue represented in the slides, and the areas of focal emphysema comprise approximately 10% of the tissue area."

Based on his review of the additional slides, Dr. Tomashefski determined that the miner suffered from simple coal workers' pneumoconiosis, but less than originally believed. Dr. Tomashefski stated that based on this new review, that he would consider the miner's coal workers' pneumoconiosis to be moderate. Dr. Tomashefski also noted that the miner had a "remote, healed, fibrocaseous granuloma, and changes in bronchi consistent with chronic bronchitis," but that was in no way attributable to coal dust exposure. Dr. Tomashefski stated that the changes of panacinar emphysema is "highly suggestive of cigarette smoking."

Dr. Tomashefski stated that since there were no pulmonary function studies available, he is unable to determine if there was any respiratory impairment as a result of the coal workers' pneumoconiosis. Dr. Tomashefski concluded that the degree of coal workers' pneumoconiosis present "would have caused, at most, very mild respiratory impairment at increased levels of exertion." This in turn would decrease the miner's ability to heavy manual labor.

*Dr. Richard L. Naeye*

Dr. Richard L. Naeye offered an opinion in this claim on September 15, 1999. (DX 22). Dr. Naeye is board certified in anatomical and clinical pathology. Dr. Naeye has also published numerous articles dealing with coal workers' pneumoconiosis.[8] Dr. Naeye reviewed the autopsy report, the medical records, and 5 slides in rendering his opinion. Dr. Naeye found a "small to moderate amount of black pigment in the lungs, adjacent to small arteries and airways and in subpleural space." Dr. Naeye also found fibrous tissue with the black pigment at some, but not all, of the sites. Dr. Naeye states that about 1 in 4 "of the black deposits has a surrounding rim of focal emphysema." Dr. Naeye also found mild to moderate centrilobular emphysema and microscopic evidence of chronic bronchitis.

Dr. Naeye interpreted his findings to show mild to moderate simple coal workers' pneumoconiosis with a moderate amount of "anthracotic macules and several micronodules." Dr. Naeye opined that there is insufficient basis for opining that the miner had any impairment in lung function that would have prevented the miner from continuing his employment as a federal mine inspector. Dr. Naeye concluded that the miner would have died at the same time and in the same manner if he had never been employed in the coal mine.

Dr. Naeye issued an additional report in this matter on March 24, 2000. (DX 35). At this time, Dr. Naeye reviewed an additional 21 slides. Dr. Naeye noted that the lung tissue in these slides was poorly preserved. Dr. Naeye found the same anthracotic macules and micronodules present. Dr. Naeye also found areas of acute lobular pneumonia of more severity than on the original slides. Dr. Naeye also found "3 intraparenchymal hyalinized nodules that range from 1-4 mm in diameter." Dr. Naeye explained that each of these nodules was surrounded with a rim of black pigment.

Dr. Naeye noted that the rim of black pigment had less dense fibrous tissue. Additionally, Dr. Naeye notes that the pigment at the edge of these nodules has a "small to moderate number of admixed birefringent crystals of all sizes." Dr. Naeye points out that there is the same microscopic evidence of chronic bronchitis and centrilobular emphysema that he had found on the original slides.

Dr. Naeye concluded that these slides do not change any of his opinions from his original report. Dr. Naeye opined that the miner suffered from mild to moderate coal workers' pneumoconiosis and a greater extent of acute lobular pneumonia than originally thought. Dr. Naeye reiterates that pneumonia was the cause of the miner's death. Again, Dr. Naeye states that the "severity of the centrilobular emphysema and chronic bronchitis raises the possibility of cigarette smoking earlier in life."

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[8] Dr. Naeye is also the chairperson of the Department of Pathology at the Pennsylvania State University College of Medicine. Dr. Naeye has also been involved in a Department of Labor committee that has examined "thousands of cases since 1979." (DX 36).

Dr. Naeye was also deposed in connection with this claim. (DX 36). Dr. Naeye explained that both black pigment and fibrous tissue must be present for there to be an impairment in lung function. Dr. Naeye also explained that the miner rapidly became incapacitated when he developed central nervous system disease. Dr. Naeye went on to discuss Dr. Tabatowski's report.

Dr. Naeye explained that Dr. Tabatowski's description of the lung surfaces as "mottled black and pink" illustrates that the disease was not severe. Dr. Naeye appears to agree with Dr. Tabatowski's description of the lung slides. Dr. Naeye stated that in the second set of slides that he examined, he found a small to moderate amount of black pigment in the lungs, and a moderate amount of fibrous tissue mixed with the black pigment in some of the lesions. Based on his microscopic findings, Dr. Naeye would not have predicted any abnormalities in the miner's lung function.

Dr. Naeye found 4 areas of anthracotic micronodules some of which had associated fibrous tissue. Dr. Naeye found that the number of coal macules and micronodules present in the miner's lungs did not cause respiratory dysfunction. Dr. Naeye also found that most of the nodules did not have any associated fibrous tissue and no significant focal emphysema. Dr. Naeye opined further that the lesions with associated fibrous tissue were too few to have caused any abnormal lung function.

Dr. Naeye stated that centrilobular emphysema is not usually caused by coal dust inhalation. However, Dr. Naeye found the cause of the miner's death to be pneumonia. Dr. Naeye also explained that pneumonia causes the affected part of the lung to not be able to properly function. Dr. Naeye opined that the miner suffered from coal workers' pneumoconiosis, pneumonia, and centrilobular emphysema. Dr. Naeye found the centrilobular emphysema to not be related to coal dust inhalation because the miner's 1980 pulmonary function study produced normal results. Dr. Naeye also found that a slight deterioration in lung function can be associated with occupational exposure to coal dust. However, this deterioration is not significant, in Dr. Naeye's opinion, because it is not progressive.

Dr. Naeye stated that the cause of the miner's death was acute lobular pneumonia with vascular disease of the brain being an indirect cause of death. Dr. Naeye found no evidence of aspiration pneumonia. Dr. Naeye opined that coal dust induced lung disease did not contribute to the miner's death nor did such disease cause any pulmonary disability in the miner. Dr. Naeye found that coal miners who mined bituminous coal have completely normal life spans. As such, simple coal workers' pneumoconiosis does not cause a compromise in lung function or Dr. Naeye would expect that the miners would not have normal life spans.

*Dr. Everett F. Oesterling, Jr.*

Dr. Everett F. Oesterling issued a report in this claim on March 17, 2000. (DX 32). Dr. Oesterling is board certified in anatomical and clinical pathology and nuclear medicine.[9] Dr. Oesterling did a microscopic description of 16 slides, the photos of which were attached to his report. Dr. Oesterling noted that based on the medical records, the miner had suffered previous strokes and had mental deterioration that had progressed as a result of cerebral vascular disease. Based on his microscopic examination of the slides, Dr. Oesterling found

> mild micronodular with macular coal workers' pneumoconiosis, the changes present in tissue appear insufficient to have significantly altered pulmonary function, thus they would not have contributed to [the miner's] death nor would they have hastened death.

In his review of the slides, Dr. Oesterling also found severe tracheobronchitis with resultant bronchopneumonia. Dr. Oesterling opined that this disease process was primarily responsible for the miner's death. Dr. Oesterling also found thromboembolic disease which Dr. Oesterling opines was a consequence of the miner's mental status. Dr. Oesterling also found moderate centrilobular pulmonary emphysema. Dr. Oesterling opines that this condition was not a result of the miner's exposure to coal dust.

Dr. Oesterling opines further that the changes caused by the emphysema were likely to have had slightly altered the miner's pulmonary function and would be responsible for the dyspnea that the miner had experienced. Dr. Oesterling concludes that the miner's

> death was the result of severe tracheobronchitis with resultant bronchopneumonia, the result of his mental status complicated by some component of thromboembolic pulmonary disease, again related to his mental status and poor hygiene. His chronic lung disease is centrilobular emphysema with areas of panlobular change which is of moderate severity. This disease process appears unrelated to mine dust, exposure. Finally, the disease due to mine dust exposure can be classified as mild to moderate micronodular with macular coal workers' pneumoconiosis. This disease has not been a factor in [the miner's] demise.

Dr. Oesterling was also deposed in connection with this claim. (EX 11). Dr. Oesterling explained that the black pigment described in the autopsy report was not necessarily indicative of

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[9] Dr. Oesterling also has 3 teaching appointments. Dr. Oesterling is the Chairperson of the Department of Pathology at Ohio Valley General Hospital.

significant coal dust exposure. Dr. Oesterling found the presence of a coal workers' pneumoconiosis nodule on the slides. Dr. Oesterling stated further that the miner had a "small nodule between the small vessel and the small airway," but that the nodule did not appear to be impeding the function of the vessel or the airway and would lead to very little alteration in the miner's pulmonary function.

Dr. Oesterling pointed out that all areas of the lung were sampled during the autopsy. Dr. Oesterling explained that on the slides, he looks for the size of the nodule and the associated scarring in the lung. Dr. Oesterling noted the presence of mild micronodular with macular coal workers' pneumoconiosis, with relatively small nodules being present. Dr. Oesterling opined that this pneumoconiosis did not affect the miner's pulmonary function. Dr. Oesterling explained that the lung has approximately 40% "reserve tissue" that must be destroyed before there will be any impairment of the lung's ability to oxygenate blood. The miner's lung did not exhibit that level of destruction.

Dr. Oesterling also discussed the miner's tracheobronchitis with resultant bronchopneumonia. Dr. Oesterling opined that the effects of these conditions were devastating to the miner. Dr. Oesterling opined that if a person were afflicted with bronchopneumonia and functionally significant coal workers' pneumoconiosis, that the person would have a more difficult time surviving. Dr. Oesterling also discussed the miner's stroke and dementia. Dr. Oesterling opines that because of these conditions, the miner was unable to swallow and cough properly and as such, was unable to cleanse his lungs properly. Dr. Oesterling explained that because the miner was bedridden, the miner was unable to clear his lungs which lead to clots being formed in the miner's lower extremities.

The fact that the miner was bedridden caused thromboembolus. Dr. Oesterling opined that this lead to a condition that caused the miner to be unable to get blood to his lungs through oxygenation. This, in turn, places an additional burden on the heart. When this is added to the embolic disease, the heart begins to fail.

Dr. Oesterling also discussed the miner's emphysema. Dr. Oesterling explained that emphysema destroys the central part of the breathing apparatus which becomes centrilobular emphysema. Dr. Oesterling states that the autopsy describes panlobular emphysema. Dr. Oesterling went on to explain that exposure to coal dust produces focal emphysema, and that centrilobular and panlobular emphysema are not caused by coal dust exposure. Dr. Oesterling stated further that centrilobular emphysema can be found in severe cases of coal workers' pneumoconiosis, but that the miner's pneumoconiosis was not severe enough to expect this to be seen.

Dr. Oesterling admitted that the miner suffered from mild to moderate centrilobular emphysema that had no measurable affect on the miner's pulmonary capacity. Dr. Oesterling opined that "not a great deal" of the miner's lung was affected by centrilobular emphysema. The amount of the lung affected would be insufficient to cause any sort of alteration in the miner's breathing. Dr. Oesterling opined that aging was the cause of the miner's emphysema and that the miner's history of pipe smoking

added to the emphysema. However, Dr. Oesterling cannot say for sure that coal dust exposure did not cause any of the miner's emphysema.

Dr. Oesterling took issue with Dr. Kahn's finding that 20-50% of the miner's lung was affected by coal workers' pneumoconiosis. Dr. Oesterling found 30% of the lung tissue to be the most significant finding. Dr. Oesterling admitted that a very limited portion of the lung had a 50% impairment showing macular changes. Dr. Oesterling opined that 40% of the terminal respiratory units would need to exhibit scarring for there to be any functional significance to the scarring. Dr. Oesterling would estimate that the amount of the lung affected would be closer to 5% of the total lung. Dr. Oesterling also took issue with Dr. Green's report.

Dr. Oesterling does not care for the usage of "severe coal workers' pneumoconiosis" in describing the extent of the miner's disease. Dr. Oesterling stated that the miner's coal workers' pneumoconiosis did not cause any functional abnormality in the miner. Dr. Oesterling stated that the miner's pneumonia and coal workers' pneumoconiosis did not work together to cause the miner's death, but that the miner's embolic condition and pneumonia worked together to produce death. Dr. Oesterling opined that all of the miner's respiratory problems at the time of his death were caused by bronchopneumonia and pulmonary emboli. Dr. Oesterling concluded that the miner's death was not contributed to in any way by coal workers' pneumoconiosis.

*Dr. Francis HY Green*

Dr. Francis HY Green issued a report regarding the miner on May 3, 2001. (CX 1). Dr. Green is a board certified in anatomic pathology.[10] Dr. Green reviewed the miner's medical records, autopsy slides, and many of the reports issued in this claim. Dr. Green found that the lung slides showed evidence of severe simple coal workers' pneumoconiosis. Dr. Green found "numerous small coal dust macules with associated focal emphysema with micronodules." Dr. Green found lesions in all of the miner's lung sections. Dr. Green also found evidence of centriacinar emphysema and chronic bronchitis. Dr. Green also found that the miner's lungs showed "necrotizing bronchopneumonia in 2 sections and an area of hemorrhage associated with pulmonary embolus." Dr. Green opined that this finding is consistent with pulmonary infarction.

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[10] Dr. Green is also a professor of pathology at the University of Calgary. Dr. Green is the chairperson of the respiratory research group at the University of Calgary. Dr. Green has two current academic appointments, has received many grants, and participates in peer review of journal articles. Dr. Green has served as a review consultant for NIOSH. Dr. Green has also written numerous journal articles most of which deal with coal workers' pneumoconiosis, coal dust exposure, and occupational lung diseases.

Dr. Green diagnosed the miner as suffering from the following conditions: severe simple coal workers' pneumoconiosis; moderately severe focal and centriacinar emphysema; chronic bronchitis; necrotizing bronchopneumonia; pulmonary embolus with infarction; and old healed infectious granulomata.

Dr. Green then rendered his opinion as to the cause of the miner's death. Dr. Green opined that the conditions that lead to the miner's death started when the miner experienced a stroke in 1995. Dr. Green also noted that the miner had been diagnosed with chronic bronchitis in 1980 and developed aspiration pneumonia in April, 1998. Dr. Green confirmed Dr. Tabatowski's report but added the observation of chronic bronchitis. Dr. Green explained that the miner died a respiratory death secondary to the complications of a previous stroke.

Dr. Green opined that the miner's "respiratory death resulted from a combination of factors including an acute necrotizing bronchopneumonia in combination with an occupational pneumoconiosis comprising simple macular coal workers' pneumoconiosis, emphysema, and chronic bronchitis." Dr. Green noted that the miner was exposed to coal dust for approximately 40 years. Dr. Green also noted that there are few chest x-rays available, and a pulmonary function study and arterial blood gas test from 1980. The blood gases were normal and the pulmonary function study showed mild obstructive impairment.

Dr. Green states that the autopsy revealed simple coal workers' pneumoconiosis, with no evidence of complicated pneumoconiosis. Dr. Green opined that the miner's severe coal workers' pneumoconiosis did not appear on the chest x-ray films because of the site of the lesions and the presence of emphysema. Dr. Green opined further that the lesions found at the autopsy occupied approximately 50% of the respiratory bronchioles. Dr. Green concluded that the miner's pneumoconiosis, emphysema, and chronic bronchitis would have combined to produce a significant obstructive ventilatory defect in the miner. Dr. Green states that there is clinical, pathological, and epidemiologic evidence that emphysema and chronic bronchitis can result from exposure to coal dust.

Dr. Green opined that the miner's death was caused by complications of the stroke and multi-infarct dementia. However, the immediate cause of the miner's death was respiratory failure due to pneumonia and coal workers' pneumoconiosis. Dr. Green opines that without any other chronic pulmonary disease, most people would survive a bout with pneumonia with appropriate treatment. Dr. Green opined that pneumoconiosis contributed to the miner's death in two ways.

First, the chronic bronchitis, which was caused by coal dust exposure, predisposed the miner to developing an infection in the respiratory tract due to accumulation of mucous and the inability to clear the miner's lungs. Secondly, pneumoconiosis contributed to the pneumonia induced hypoxemia experienced by the miner by reducing the oxygen flow from the air into the miner's blood stream. Dr. Green concluded that the miner died from respiratory complications of the stroke. Respiratory failure

was due to pneumonia and coal workers' pneumoconiosis. Dr. Green opines that such coal workers' pneumoconiosis was a major contributing factor in the miner's death.

*Dr. Perry Guariglia*

Dr. Perry Guariglia issued a report in connection with this claim. (CX 2). Dr. Guariglia is board certified in anatomical and clinical pathology.[11] Dr. Guariglia reviewed and summarized multiple records, reviewed 24 lung tissue slides, and reports in this record. Dr. Guariglia noted that the miner worked for 20 years in an underground coal mine and 20 years as a federal mine inspector. Dr. Guariglia also noted that the miner never smoked.

Dr. Guariglia opined that the miner suffered from "acute, necrotizing bronchopneumonia; moderate centrilobular emphysema; coal workers' pneumoconiosis with coal dust macules and silica; silicotic nodules; and pulmonary thromboemboli." Dr. Guariglia also found that the miner suffered from several non-pulmonary conditions that included mild coronary artherosclerosis, a history of strokes, and dementia.

Dr. Guariglia concluded that it was "highly probable" that the miner's centrilobular emphysema was caused by the miner's exposure to coal dust. Dr. Guariglia bases this finding on the fact that the miner was a non-smoker and the medical evidence that has established that centrilobular emphysema can be caused by exposure to coal dust. Dr. Guariglia's examination of the lung tissue slides found numerous coal macules "adjacent to and within many areas of emphysema." Dr. Guariglia also found coal macules and emphysema within the areas of acute bronchopneumonia.

Dr. Guariglia opined further that the miner's emphysema and coal workers' pneumoconiosis combined to contribute to the miner's development of acute bronchopneumonia. Dr. Guariglia concluded that, based on the pulmonary pathology, that it is more likely that the acute bronchopneumonia was precipitated by the miner's coal workers' pneumoconiosis and emphysema. Dr. Guariglia determined that the miner's direct cause of death was "cardiorespiratory failure due to acute, necrotizing bronchopneumonia, and that bronchopneumonia was brought on, at least in substantial part, by emphysema which was related to occupational coal dust exposure, as well as 'classical' coal workers' pneumoconiosis."

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[11] Dr. Guariglia also has a subspecialty certification in cytopathology. Dr. Guariglia is an instructor of pathology at Rush Medical College. Dr. Guariglia possesses 5 current appointments and 6 non-active appointments.

*Dr. Ben V. Branscomb*

Dr. Ben V. Branscomb issued a report in the claim on January 17, 2001. (EX 1). Dr. Branscomb is board certified in internal medicine and pulmonary disease.[12] (EX 2). Dr. Branscomb noted that the miner was exposed to coal dust for approximately 40 years. Dr. Branscomb concluded that such exposure would be sufficient for a person to develop coal workers' pneumoconiosis. Dr. Branscomb also noted that the miner did not have a cigarette smoking history but that pipe smoking had been noted. Dr. Branscomb reviewed and outlined the miner's history as it was listed in the different reports. Dr. Branscomb also noted the miner's 1980 pulmonary function study and arterial blood gas test. Dr. Branscomb recognized that the pulmonary function study produced results showing minimal obstructive changes. Dr. Branscomb noted that the miner's 1980 arterial blood gas test showed no indication of any blood gas impairment.

This information lead Dr. Branscomb to conclude that "insufficient objective evidence" existed to justify a diagnosis of coal workers' pneumoconiosis. Additionally, Dr. Branscomb found that there was no evidence nor any basis for finding that the miner suffered any pulmonary impairment. Dr. Branscomb bases this conclusion on the fact that the miner's testing showed normal function levels and that the tests could not be validated, in addition to the miner's severe neurological problems and pathologic changes of coal workers' pneumoconiosis or emphysema would not be expected to produce shortness of breath.

Dr. Branscomb also concluded that the miner possessed the pulmonary capacity to perform his last coal mine employment prior to suffering multiple strokes and the onset of dementia. Dr. Branscomb concluded that neither coal workers' pneumoconiosis nor any other coal dust induced condition had any effect on the "timing or circumstances of [the miner's] death."

Dr. Branscomb issued a supplemental report on January 25, 2001, after reviewing the deposition testimony of Dr. Kahn. (EX 3). Dr. Branscomb's opinion of January 17, 2001 remained unchanged. Dr. Branscomb took issue with Dr. Kahn's finding that lung tissue samples are usually representative of the lung as a whole. Dr. Branscomb believes that this is not the usual practice of pathologists and that the samples usually show only the most diseased parts of the lungs. Dr. Branscomb also took issue with Dr. Kahn's determination that once nodules develop that there is a corresponding loss in pulmonary reserve. Dr. Branscomb is of the opinion that simple coal workers' pneumoconiosis can be present without the presence of a decline in pulmonary function.

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[12] Dr. Branscomb is a professor of medicine in emphysema and respiratory disease. Dr. Branscomb is also the Associate Director of Respiratory Therapy at the University of Alabama at Birmingham Hospitals and Clinics. Dr. Branscomb is a member of several consultative staffs and has conducted numerous lectures. (EX 2). Dr. Branscomb stated that he was a part of the National Heart Institute for 2 years and helped to develop the lung program as a part of that institute. (EX 5).

Dr. Branscomb concluded that the miner "could not have lived a half an hour longer than he did nor would his terminal months or years or illness have been in any way different had he never been a coal miner." Dr. Branscomb opines that the miner died from neurovascular disease. Dr. Branscomb was also deposed in connection with this claim. (EX 5).

Dr. Branscomb testified that he reviewed a "thorough and ample series of records." Dr. Branscomb believes that he is in the best position to render an opinion in this claim because he has the opportunity to review the reports of the consulting physicians. Dr. Branscomb reiterated that insufficient medical data exists to suspect that the miner developed a coal dust induced lung disease. Dr. Branscomb bases this on the available chest x-ray evidence, the miner's symptoms, and the clinical findings regarding the miner. Dr. Branscomb pointed out that he would expect there to be some reference to pulmonary problems in the documentation regarding the miner's stroke.

Dr. Branscomb acknowledges that coal workers' pneumoconiosis was found at the miner's autopsy. Dr. Branscomb then discussed the miner's complaints of cough and shortness of breath during his lifetime. Dr. Branscomb opined that neither of these symptoms indicated chronic lung disease. Dr. Branscomb noted that the miner suffered from a variety of chronic conditions which include hypertension, gastrointestinal bleeding, alcoholism, severe psychotic disturbances, dementia, paralysis multiple small strokes, renal disease, and the inability to speak or swallow. Dr. Branscomb opined that these conditions are part of a pattern of vascular disease and hypertension.

Dr. Branscomb then discussed the reports issued by several other physicians in this claim. Dr. Branscomb disagreed with the opinion of Dr. Ulrich. Dr. Branscomb does not believe that the miner possessed an already weakened lung condition. Dr. Branscomb bases this opinion on the fact that a previously weakened condition was not mentioned by any other doctors or in any of the miner's treatment plans. Dr. Branscomb opined further that considering the miner's neurological problems and swallowing problems that it is "unreasonable to think that anything would increase [the miner's] chances of survival." Dr. Branscomb elaborated that the miner had a lot of severe problems all of which culminated resulted in the terminal event and made survival impossible.

Dr. Branscomb also took issue with the report of Dr. Kahn. Dr. Branscomb states that the lesions in the miner's lungs were not large enough to produce an affect on the miner's ventilation diffusion or profusion. Dr. Branscomb bases this conclusion on the fact that if 30% or 50% of the miner's terminal respiratory units were nonfunctional, as Dr. Kahn opined, then there would be a substantial decrease in the miner's pulmonary function. Dr. Branscomb stated that in the majority of persons with simple coal workers' pneumoconiosis, that he usually sees normal pulmonary function.

Dr. Branscomb also stated that the slides showed insufficient evidence to have caused any of the described impairments. However, Dr. Branscomb admitted that he had never examined the lung tissue slides. Dr. Branscomb stated that if Dr. Kahn's conclusions are correct, then there is insufficient evidence to establish the presence of significant coal workers' pneumoconiosis that would decrease the

miner's pulmonary capacity. Dr. Branscomb stated that it is possible to assess pulmonary function by looking at the scarring present on the lung slides. Dr. Branscomb states that the scarring that was described would not be sufficient to result in restriction.

Dr. Branscomb also discussed the miner's emphysema and aspiration pneumonia. Dr. Branscomb opined that aspiration pneumonia can occur once a person suffers a stroke and that usually causes the person to not live much longer. Dr. Branscomb opined further that the miner had terminal pneumonia. Dr. Branscomb concluded that the miner suffered from an impairment and sequelae related to vascular disease which was severe enough to have anticipated the miner's death.

Dr. Branscomb testified that coal workers' pneumoconiosis did not in any way hasten the miner's death. Dr. Branscomb stated that the miner would have likely recovered from the pneumonia had the miner not experienced the previous strokes. Dr. Branscomb opined that the level of coal workers' pneumoconiosis present in the miner was not enough to decrease the miner's pulmonary reserve. Dr. Branscomb bases this conclusion, in part, on the fact that there is no evidence in the records of any significant progressive lung disease.

Dr. Branscomb issued a second supplemental report on June 4, 2001. (EX 10). At that time, Dr. Branscomb had reviewed the report of Dr. Green. Dr. Branscomb states that Dr. Green found more numerous lesions than had been found by the other pathologists. Dr. Branscomb also found no convincing evidence to establish that the miner suffered from significant chronic bronchitis. Dr. Branscomb opined that "to conclude that the current bouts of aspiration pneumonia killed [the miner] with recurrent strokes is an exceedingly straightforward conclusion based on the medical records."

Dr. Branscomb further opined that the pneumoconiosis found on the slides by Dr. Green would not have caused any pulmonary function or airway patency impairment. Dr. Branscomb states that it would be speculative to say that the miner's coal workers' pneumoconiosis would produce respiratory failure. Dr. Branscomb explained that pneumonia is the most common cause of death in people who have suffered recurrent strokes and chronic aspiration. Dr. Branscomb disagrees with Dr. Green that coal workers' pneumoconiosis was a major contributor to the miner's death. Dr. Branscomb would describe the coal workers' pneumoconiosis as an incidental finding in the miner's autopsy.

Dr. Branscomb also took issue with Dr. Guariglia's findings. Dr. Branscomb stated that there is no correlation of the miner's overall pulmonary function with the emphysema present on the slides that were sampled to "show disease." Dr. Branscomb also states that Dr. Guariglia fails to mention that the pneumonia found in the miner is not the pattern usually associated with coal workers' pneumoconiosis nor emphysema.

Dr. Branscomb then took issue with Dr. Cohen. Dr. Branscomb does not believe that the miner's emphysema and chronic bronchitis resulted in a substantial pulmonary impairment. Dr.

Branscomb points out that the pulmonary function study conducted in 1980 was invalidated and that the results of that test were not sufficient to produce significant symptoms.

Dr. Branscomb concluded that there is no "reasonable medical basis for assuming that significant chronic obstructive pulmonary disease and impairment were present."

*Dr. Robert Cohen, Jr.*

Dr. Robert Cohen reviewed various data, histories and physicals, hospital records, the death certificate, and physician reports and rendered an opinion in this claim. (CX 3). Dr. Cohen is board certified in internal medicine, critical care and pulmonary disease.[13] Dr. Cohen noted that the miner was engaged in coal mine employment for 19 to 20 years and served as a federal mine inspector for 20 years. Dr. Cohen also noted that the miner did not smoke cigarettes.

Dr. Cohen stated that, based on his review of the record, the miner suffered from coal workers' pneumoconiosis. Dr. Cohen found this condition to be substantially related to the miner's coal mine employment and "modest" smoking of a pipe. Dr. Cohen determined that the history of pipe smoking may have contributed to the miner's chronic respiratory condition.

Dr. Cohen explained that the miner's exposure to coal dust would be considered significant. The miner worked for 20 years in underground coal mine employment, all of which, Dr. Cohen points out, occurred before the imposition of federal dust control regulation. Dr. Cohen also takes into consideration the fact that the miner was employed for 20 years as a federal mine inspector which exposed the miner to underground coal mine conditions.

Dr. Cohen noted that two examining physicians and several consulting physicians noted symptoms of chronic lung disease. Dr. Cohen stated that the miner had a pulmonary function study 18 years before the miner's death showing the onset of mild obstructive lung disease. Dr. Cohen also discussed the miner's obstructive lung disease and coal dust exposure. Dr. Cohen explained that the miner suffered from obstructive lung disease, more specifically severe focal and centrilobular emphysema and chronic bronchitis. Dr. Cohen bases these findings on the miner's clinical history and the mild impairment present on the one pulmonary function study.

Dr. Cohen went on to explain that obstructive defects can occur from coal dust exposure. Dr. Cohen states that medical data shows a relationship between developing obstructive impairments,

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[13] Dr. Cohen also reviews articles for peer review journals and has published numerous articles. Dr. Cohen also currently has 8 appointments and has spoken numerous times regarding occupational pneumoconiosis. Dr. Cohen is a NIOSH certified B-Reader.

which manifest as chronic bronchitis or emphysema and coal dust inhalation. Dr. Cohen reviewed the literature that supports this conclusion.

Dr. Cohen then turned his attention to the facts specific to this particular miner. Dr. Cohen states that the miner suffered from obstructive lung disease based on the miner's history and physiologic testing conducted in 1980. Dr. Cohen went on to state that lung damage progresses after the miner ceased to be exposed to coal dust. Dr. Cohen also notes that 20-50% of the miner's terminal respiratory units were involved with the pathologic changes of interstitial lung disease due to the miner's coal workers' pneumoconiosis. Dr. Cohen went on to explain that the miner suffered from emphysema caused by coal dust exposure.

Dr. Cohen noted that the miner's emphysema and interstitial lung disease caused a "significant decrease in the ability of [the miner's] lung to withstand additional respiratory compromise." Dr. Cohen explained that persons with an underlying pulmonary disease cannot withstand further pulmonary injury and will succumb sooner. Dr. Cohen took issue with Dr. Naeye's finding that pneumonia does not contribute to a respiratory death. Dr. Cohen states that pneumonia "causes severe derangement in gas exchange and patients, especially those patients with pre-existing lung disease are at greater risk of dying from additional impairment."

All of this information, taken together, lead Dr. Cohen to conclude that the miner's exposure to coal dust lead to the development of coal workers' pneumoconiosis, emphysema, and chronic bronchitis. Dr. Cohen believes that the miner's history of pipe smoking also significantly contributed to the development of emphysema and chronic bronchitis. Dr. Cohen opines that these impairments lead to a substantial pulmonary impairment. Because of this impairment, Dr. Cohen believes that the miner was not able to withstand the effects of pneumonia and pulmonary emboli and that the miner died sooner than he would have if he was not afflicted with coal workers' pneumoconiosis.

*Dr. Gregory Fino*

Dr. Gregory Fino reviewed the medical records in this claim and issued a report on his findings on June 13, 2001. (EX 9). Dr. Fino is board certified in internal medicine and pulmonary disease.[14] Dr. Fino noted that the miner was engaged in coal mine employment for 42 years. Dr. Fino reviewed the medical documentation and noted that the miner's autopsy was limited to the lungs. Dr. Fino found that this limitation rendered him unable to determine a cause of death. Dr. Fino went on to state that it would be "speculative for anyone to state that lung disease contributed to the cause of death because [one] does not know if [the miner] suffered some other traumatic event." Dr. Fino also states that it is

[14] Dr. Fino is also a NIOSH certified B-Reader. Dr. Fino is an associate clinical professor of medicine and pulmonary disease at the University of Pittsburgh Medical School. Dr. Fino has also published numerous articles regarding occupational lung diseases. (EX 12).

"pure speculation to say that coal workers' pneumoconiosis was a contributing cause of [the miner's] death."

Dr. Fino, however, did agree with the pathologists that there is evidence that coal workers' pneumoconiosis existed in this miner. Dr. Fino noted that there is no evidence of any chronic hypoxia. Dr. Fino also noted that the miner suffered from dementia due to multiple strokes. Dr. Fino opined that this condition put the miner at risk for developing blood clots in his lungs and pneumonia. Dr. Fino points out that coal workers' pneumoconiosis does not cause any of these diseases to occur with any more frequency.

Dr. Fino states that even if he assumes that the miner suffered from progressive respiratory failure due to aspiration, that the pneumonia was caused by the previous stroke and the blood clots in the miner's lungs. Dr. Fino points out that none of these conditions are caused by coal dust exposure. Dr. Fino concludes that the miner "would have died at the same time and in the same way" had he never been exposed to coal dust. Dr. Fino also concludes that

> those saying that coal workers' pneumoconiosis was a contributing cause [to the miner's death] have no valid objective evidence to support that claim. The mere finding of pathologic pneumoconiosis does not imply pulmonary impairment or any pulmonary limitation. Furthermore, there is no objective evidence in the medical record of any respiratory impairment or pulmonary disability.

All of this lead Dr. Fino to make the following conclusions: the miner had pathologic evidence of coal workers' pneumoconiosis; there was no evidence of any respiratory impairment; that the exact cause of death cannot be determined because a full autopsy was not performed; that even if assuming that the miner suffered from respiratory failure, Dr. Fino is confident that such respiratory failure was not caused by, contributed to, nor hastened by coal dust exposure; and that the objective medical data does not show that the miner's death caused by, contributed to, nor hastened by the inhalation coal dust.

*Good Samaritan Hospital Records*

Included in the record are various medical documents from Good Samaritan Hospital. (DX 9). The miner was admitted to the hospital on November 21, 1995. At that time, the admitting physician noted that the miner's lungs sounds were chronically diminished. It was also noted that the miner was very reluctant to seek medical treatment. The admission note for this visit indicates that the miner's lungs showed an increased AP diameter indicating probable chronic obstructive pulmonary disease. The admission document notes that the miner has a history of coal mine employment.

The miner was again admitted to this hospital on February 10, 1997. At that time, it was noted that the miner's lung sounds were diminished. An admission consultation was conducted at this time by Dr. John P. Feerick. Dr. Feerick makes no note of any respiratory condition. The miner was a patient at this hospital again from October 16, 1997 through October 19, 1997. The miner was treated for a non-respiratory condition. The miner was again admitted to Good Samaritan Hospital from April 23, 1998 to May 1, 1998. The documentation for this stay noted that the miner suffered a stroke, suffered from multi-infarct dementia, and contracted pneumonia. It was noted that the miner's lungs were "congested with some scattered loose rhonci and extremely poor and weak cough."

*Bethesda Hospital Records*

The records from Bethesda Hospital in this claim span from February 6, 1997 through May 1, 1998. (DX 37). The miner was admitted to Bethesda on February 6, 1997 for a left knee replacement. The miner was again a patient at Bethesda from October 16, 1997 to October 19, 1997. There is no mention of any pulmonary problems in either of these visits. The miner was treated at the emergency room at Bethesda on April 23, 1998. At that time, the miner's lungs showed diminished breath sounds with no wheezing or rales. However, on the miner's history and physical for this same visit, the miner's lungs were noted to be clear bilaterally. The miner was hospitalized from April 23, 1998 to May 1, 1998. The miner was, at that time, diagnosed as suffering from pneumonia, among other conditions. The miner's lungs were noted to be "moderately congested with some scattered loose rhonci and extremely poor and weak cough."

*Physician Report of Occupational Pneumoconiosis*

Included in the record in this claim is a report from the West Virginia Workers' Compensation Fund. (DX 10). The physician's name affixed to this report is illegible. This document notes that the miner suffered from shortness of breath, and an increase in both sputum and cough. The report also notes that the miner was exposed to coal dust for 40 years and was a non-smoker. The miner was diagnosed as suffering from coal workers' pneumoconiosis.

*Affidavits*

*Jack A. Cologie*

Jack A. Cologie submitted an affidavit in this claim. (CX 5). Mr. Cologie stated that he was the miner's direct supervisor from 1981 to 1990 when the miner was employed as a federal coal mine inspector. Mr. Cologie stated further that the miner spent 85-90% of his total work time inside an operating underground coal mine. Mr. Cologie pointed out that the miner was present in all areas of the mine, including the face of the mine. Mr. Cologie explained that the other time that the miner was at work, he was engaged in activities at strip mines, surface areas of underground coal mines, and meetings that occurred away from any coal mine site. Mr. Cologie pointed out that the majority of

mines that the miner entered as part of his employment were on "reduced dust standard." Mr. Cologie also stated that he had never seen the miner smoke a cigarette.

*John Zavora*

John Zavora also submitted an affidavit in this claim. (CX 6). Mr. Zavora stated he was the miner's supervisor from 1971 to 1981. Mr. Zavora explained that the miner's duties as a federal coal mine inspector included being inside the underground coal mines inspecting all areas of the mines. Mr. Zavora explained that this exposed the miner to "substantial amounts of coal dust and rock dust." Mr. Zavora estimated that the miner spent approximately 50% of his time at operating underground coal mine sites that "were in violation of the standard for respirable coal dust." Mr. Zavora also stated that he had never seen the miner smoke a cigarette.

## Discussion and Analysis

## Responsible Operator

Employer has contested its designation as the proper responsible operator in this claim. Employer raises this issued based on the fact that the miner was last employed in the coal mine industry by the United States Department of Labor, Mine Safety and Health Administration ("MSHA") as a mine inspector. It is undisputed that the miner worked for approximately 20 years as a federal mine inspector. Employer contends that if liability is to be assessed for benefits under the Act, that MSHA should assume the payment of those benefits.

In *Moore v. Duquesne Light Co.*, 4 B.L.R. 1-40 (1981), the Benefits Review Board ("Board") reasoned that the Department of Interior's Bureau of Mines, which was operating a coal mine, could be considered an operator. However, the Board also held that the United States is not legally capable, for purposes of the regulations, of providing benefits to a claimant. Civil liability may not be imposed upon the sovereign except to the extent and in the manner to which it has consented. *Dalehite v. United States,* 346 U.S. 15 (1953). In the Federal Employee's Compensation Act, Congress provided a federal employee's exclusive cause of action against the United States, and therefore, the Bureau of Mines is incapable of assuming any liability for payment under the Black Lung Benefits Act. Although the Board in *Spradlin v. Island Creek Coal Co.*, 6 B.L.R. 1-716 (1984), refused to address whether the Mine Enforcement Safety Administration could be found to be a responsible operator, it is logical that the same analysis would apply.

Additionally, Congress has decided to make explicit that

> [n]either the United States, nor any State, nor instrumentality or agency of the United States or any State, shall be considered an operator.

29 C.F.R. §725.491(f). Therefore, it is inappropriate for this Court to designate the United States Department of Labor, Mine Safety and Health Administration as the responsible operator in this claim.

Employer, Consol Inc., has not disputed that it employed the miner for the time prior to his employment as a federal mine inspector. As such, I find that Consol Inc. is the properly designated responsible operator in this claim and will be responsible for the payment of any benefits found due to claimant.

**Death Due to Pneumoconiosis**

Considering the fact that the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c).

Section 718.205, states, in pertinent part:

> (c) For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at § 718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where a miner's death was caused by a traumatic injury or principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.
>
> (5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

Claimant's entitlement to benefits on her survivor's claim is dependent upon her meeting the burden of establishing that the miner's death was due to coal workers' pneumoconiosis. 20 C.F.R. § 718.205. Claimant is able to meet this burden by establishing any of the elements of § 718.205(c).

The record contains no evidence of complicated pneumoconiosis which would enable the claimant to invoke the presumption at Section 718.304. Therefore, the miner's death was not due to pneumoconiosis pursuant to § 718.205(c)(3).

There is no competent medical evidence contained in the record to establish that pneumoconiosis was the cause of the miner's death. As such, claimant has failed to establish that the miners death was due to pneumoconiosis pursuant to § 718.205(c)(1).

There remains only one way for claimant to establish that the miner's death was due to pneumoconiosis. Pursuant to § 718.205(c)(2), claimant can establish that the miner's death was due to pneumoconiosis by providing evidence that establishes that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or that the miner's death was caused by complications of pneumoconiosis. The physicians of record disagree as to whether the miner's death was contributed to by his pneumoconiosis.

As a preliminary issue, I find that the miner was not a cigarette smoker in his lifetime. This finding is supported by the affidavits of two of the miner's coworkers (CX 5&6) and the testimony of claimant. Additionally, there has been considerable discussion of the practice of pathologists when taking samples of lungs at autopsy. I find that the miner's lungs were represented by the slides taken. Dr. Tabatowski noted in the autopsy report that all of miner's lobes were sampled. Therefore, I find that the slides were not taken to represent only the diseased areas of the miner's lungs.

I accord little weight to the report from the West Virginia Workers' Compensation Fund. The physician certifying this document cannot be determined. Additionally, it offers no reasoning for the finding. Therefore, I accord little weight to the Report of Occupational Pneumoconiosis from the West Virginia Workers' Compensation Fund. Additionally, I accord less weight to the autopsy report of Dr. Tabatowski. Dr. Tabatowski found the presence of pneumoconiosis at the autopsy. However, the physician does not render any opinion as to whether or not this condition contributed to the miner's death. As such, it is accorded less weight.

Eleven physicians rendered opinions in this claim. Five of the physicians found that pneumoconiosis had nothing to do with the miner's death, the other five finding that pneumoconiosis substantially contributed to the miner's death. Drs. Tomashefski, Naeye, Oesterling, Branscomb, and Fino all found that pneumoconiosis did not contribute to the miner's death. Dr. Tomashefski found that the miner suffered from "moderate" pneumoconiosis that did not contribute to the miner's death. Dr. Tomashefski also found that simple coal workers' pneumoconiosis "almost never" causes death. Dr. Tomashefski found that the miner's condition was "highly suggestive" of a cigarette smoking history. I find Dr. Tomashefski's opinion entitled to less weight. Dr. Tomashefski determined that the miner's coal workers' pneumoconiosis did not contribute to his death, yet offers no explanation for the miner's multiple pulmonary conditions other than to attribute them to cigarette smoking. It is clear from the overwhelming evidence submitted that the miner was a non-smoker. Therefore, I find that Dr.

Tomashefski's opinion is less well reasoned than the other physician reports of record and thus entitled to less weight.

Dr. Naeye also found that the miner suffered from mild to moderate coal workers' pneumoconiosis. However, Dr. Naeye also found that the miner possibly was a cigarette smoker in life based on the findings of centrilobular emphysema and chronic bronchitis. Dr. Naeye bases the conclusion that the miner's centrilobular emphysema was not caused by coal dust because the miner had a normal pulmonary function study in 1980. I find this not to be well reasoned. Therefore, I find Dr. Naeye's opinion entitled to less weight.

Drs. Oesterling and Branscomb both submitted well reasoned and well documented reports in this claim. I find that both of these physicians are well qualified and I accord great weight to their opinions. I accord less weight to the opinion of Dr. Fino. Dr. Fino opines that it would be speculative to determine a cause of death in this claim because a full autopsy was not performed. Dr. Fino concludes that even if the miner suffered a respiratory death, it was not caused or contributed to by coal dust exposure. I find Dr. Fino's report to not be well reasoned. This is so because Dr. Fino offers no explanation for his conclusion. Additionally, Dr. Fino's determination that specifying a cause of death would be speculative runs contrary to all of the other physician reports contained in the record. Therefore, I find Dr. Fino's report entitled to less weight.

Drs. Ulrich, Kahn, Green, Guariglia, and Cohen all determined that coal workers' pneumoconiosis substantially contributed to the miner's death. Dr. Ulrich determined that the miner died more quickly from the pneumonia than he would have if he did not suffer from a chronic respiratory disease. Dr. Ulrich bases his conclusion on his observations of the miner and the miner's shortness of breath just before the miner's death. I find Dr. Ulrich's opinion entitled to less weight than the other opinions of record. This is so because Dr. Ulrich's observations of the miner were very limited because of the miner's reluctance to seek medical treatment. Additionally, Dr. Ulrich did not review the autopsy slides in this claim.

Dr. Kahn found that the miner's terminal respiratory unites were involved with coal workers' pneumoconiosis macules. Dr. Kahn also found that chronic lung disease cause the lung to be unable to overcome insults. Because of this, Dr. Kahn determined that coal workers' pneumoconiosis contributed to the miner's death. Dr. Guariglia found that the miner suffered from not only coal workers' pneumoconiosis, but also centrilobular emphysema that was the result of exposure to coal dust. Dr. Kahn found that these two conditions combined to contribute to the miner's acute bronchopneumonia that ultimately lead to the miner's demise. I find the opinions of Dr. Kahn and Dr. Guariglia to be well reasoned and based on the objective medical evidence contained in the record. Accordingly, I accord more weight to the opinions of these physicians.

Dr. Green found that the miner suffered from severe pneumoconiosis which combined with several other factors to produce a respiratory death. Dr. Green found that pneumoconiosis contributed

to the miner's death by predisposing the miner to developing pneumonia. Dr. Cohen found that the miner suffered from coal workers' pneumoconiosis involving 20-50% of the terminal respiratory units. Dr. Cohen found that the miner suffered from emphysema due to coal dust exposure that left the miner unable to withstand any further compromise to his lungs. Dr. Cohen determined that the miner died sooner because of pneumoconiosis. I find the opinions of Drs. Green and Cohen to be more well reasoned and entitled to more weight.

Weighing all of the physician opinion evidence, I find that claimant has established her burden pursuant to § 718.205(c)(2). I find the opinions fo Dr. Oesterling and Branscomb to be very well reasoned. However, those opinions are outweighed by the opinions of Drs. Kahn, Green, Guariglia, and Cohen, all of which are also well reasoned and well supported by the evidence of record. Accordingly, I find that claimant has established that the miner's death was substantially contributed to by pneumoconiosis or that death was caused by the complications of pneumoconiosis.

**Entitlement**

As claimant has established that the miner's death was due to pneumoconiosis under the provisions of Section 718.205(c)(2), she is entitled to survivor's benefits under the Act for the time period in which she remains a qualifying widow under the Act.

**Attorney Fee**

An application by the claimant's attorney for approval of a fee has not been received and, therefore, no award of attorney's fees for services is made. Thirty days is hereby allowed to claimant's counsel for the submission of such an application and attention is directed to Sections 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties, including claimant, must accompany the application. Parties have ten days following the receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

**ORDER**

IT IS HEREBY ORDERED that the claim of Kathryn L. Cornett, survivor of Elmer R. Cornett for benefits under the Act is GRANTED.

IT IS FURTHER ORDERED that Kathryn L. Cornett be paid all benefits to which she is entitled commencing on May 1, 1998.

A
ROBERT J. LESNICK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this (Order of Dismissal/Decision and Order) may appeal it to the Benefits Review Board within 30 days from the date of this (Order of Dismissal/Decision and Order), by filing a notice of appeal with the ***Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601***. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.